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NEWS 2	JUL 28	CA/CAplus patent coverage enhanced
NEWS 3	JUL 28	EPFULL enhanced with additional legal status information from the epoline Register
NEWS 4	JUL 28	IFICDB, IFIPAT, and IFIUDB reloaded with enhancements
NEWS 5	JUL 28	STN Viewer performance improved
NEWS 6	AUG 01	INPADOCDB and INPAFAMDB coverage enhanced
NEWS 7	AUG 13	CA/CAplus enhanced with printed Chemical Abstracts page images from 1967-1998
NEWS 8	AUG 15	CAOLD to be discontinued on December 31, 2008
NEWS 9	AUG 15	CAplus currency for Korean patents enhanced
NEWS 10	AUG 27	CAS definition of basic patents expanded to ensure comprehensive access to substance and sequence information
NEWS 11	SEP 18	Support for STN Express, Versions 6.01 and earlier, to be discontinued
NEWS 12	SEP 25	CA/CAplus current-awareness alert options enhanced to accommodate supplemental CAS indexing of exemplified prophetic substances
NEWS 13	SEP 26	WPIDS, WPINDEX, and WPIX coverage of Chinese and and Korean patents enhanced
NEWS 14	SEP 29	IFICLS enhanced with new super search field
NEWS 15	SEP 29	EMBASE and EMBAL enhanced with new search and display fields
NEWS 16	SEP 30	CAS patent coverage enhanced to include exemplified prophetic substances identified in new Japanese-language patents
NEWS 17	OCT 07	EPFULL enhanced with full implementation of EPC2000
NEWS 18	OCT 07	Multiple databases enhanced for more flexible patent number searching
NEWS 19	OCT 22	Current-awareness alert (SDI) setup and editing enhanced
NEWS 20	OCT 22	WPIDS, WPINDEX, and WPIX enhanced with Canadian PCT Applications
NEWS 21	OCT 24	CHEMLIST enhanced with intermediate list of pre-registered REACH substances

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ISOFORM 1)/CN

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L1           1 96720-06-8/RN

=> DIS L1 1 SQIDE

L1   ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2008 ACS on STN
RN   96720-06-8  REGISTRY
CN   3,5,8-Trioxa-4-phosphahexacos-17-en-1-aminium, 4-hydroxy-N,N,N-trimethyl-,
     inner salt 4-oxide  (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN   3,5,8-Trioxa-4-phosphahexacos-17-en-1-aminium, 4-hydroxy-N,N,N-trimethyl-,
     inner salt, 4-oxide (9CI)
MF   C25 H52 N O5 P
LC   STN Files:   BEILSTEIN*, CA, CAPLUS, CASREACT, CHEMCATS, CSCHEM, MEDLINE,
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RL.NP  Roles from non-patents:  PREP (Preparation)
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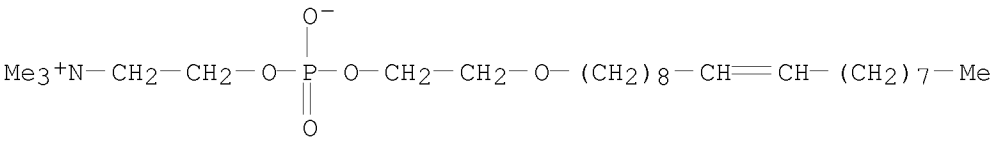

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L2   ANSWER 1 OF 27     MEDLINE on STN
ACCESSION NUMBER:   2004505822     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 15474031
TITLE:              Interactions of 12-lipoxygenase with phospholipase A2
                    isoforms following platelet activation through the
                    glycoprotein VI collagen receptor.
AUTHOR:             Coffey Marcus J; Coles Barbara; Locke Matthew;
                    Bermudez-Fajardo Alexandra; Williams P Claire; Jarvis Gavin
                    E; O'donnell Valerie B
CORPORATE SOURCE:   Department of Medical Biochemistry and Immunology, Wales
                    College of Medicine, Cardiff University, Heath Park,
                    Cardiff CF14 4XN, UK.. coffeymj@cardiff.ac.uk
SOURCE:             FEBS letters, (2004 Oct 8) Vol. 576, No. 1-2, pp. 165-8.
                    Journal code: 0155157. ISSN: 0014-5793.
PUB. COUNTRY:       Netherlands
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
                    (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
ENTRY MONTH:        200411
ENTRY DATE:         Entered STN: 13 Oct 2004
                    Last Updated on STN: 19 Dec 2004
                    Entered Medline: 19 Nov 2004
AB   Recent studies implicate the collagen receptor, glycoprotein VI (GPVI) in
     activation of platelet 12-lipoxygenase (p12-LOX).  Herein, we show that
     GPVI-stimulated 12-hydro(peroxy)eicosatetraenoic acid (H(P)ETE) synthesis
     is inhibited by palmityl trifluromethyl ketone or
     oleyloxyethylphosphocholine , but not bromoenol lactone,
     implicating secretory and cytosolic, but not calcium-independent
     phospholipase A2 (PLA2) isoforms.  Also, following GPVI activation, 12-LOX
     co-immunoprecipitates with both cytosolic and secretory PLA2 (sPLA2).
     Finally, venoms containing sPLA2 acutely activate p12-LOX in a
     dose-dependent manner.  This study shows that platelet 12-H(P)ETE
     generation utilizes arachidonate substrate from both c- and sPLA2 and that
     12-LOX functionally associates with both PLA2 isoforms.
     Copyright 2004 Federation of European Biochemical Societies

L2   ANSWER 2 OF 27     MEDLINE on STN
ACCESSION NUMBER:   2004455640     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 15364285
TITLE:              Phospholipase A2 in salivary glands isolated from tobacco
                    hornworms, Manduca sexta.
AUTHOR:             Tunaz Hasan; Stanley David W
CORPORATE SOURCE:   Insect Biochemical Physiology Laboratory, University of
                    Nebraska-Lincoln, 311 Plant Industry Building, Lincoln, NE
                    68583-0816, USA.
SOURCE:             Comparative biochemistry and physiology. Part B,
                    Biochemistry & molecular biology, (2004 Sep) Vol. 139, No.
                    1, pp. 27-33.
                    Journal code: 9516061. ISSN: 1096-4959.
PUB. COUNTRY:       England: United Kingdom
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
                    (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
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ENTRY MONTH: 200506
ENTRY DATE: Entered STN: 15 Sep 2004
Last Updated on STN: 1 Jul 2005
Entered Medline: 30 Jun 2005

AB We describe a phospholipase A2 (PLA2) associated with the salivary glands of tobacco hornworms, Manduca sexta. This enzyme is able to hydrolyze arachidonic acid from the sn-2 position of PLs. Addition of the calcium chelator, EGTA, or calcium, to the Tris reaction buffer impaired the PLA2 activity, from which we infer the enzyme requires very low concentrations of calcium or perhaps other ions for optimal activity. PLA2 activity was sensitive to protein concentration (highest activity at 25 microg protein per microl), reaction time (optimal at 30 min), buffer pH (optimal at pH 8-10), and reaction temperature (optimal range 18-38 degrees C). The salivary gland PLA2 was sensitive to the site-specific inhibitor, oleyloxyethylphosphorylcholine and stable to freezing at -80 degrees C, but not -20 degrees C. The biological significance of this enzyme may relate to hydrolysis of fatty acid moieties from dietary PLs for absorption by midgut epithelia. This salivary gland enzyme may also be responsible for killing food-borne bacteria.

L2 ANSWER 3 OF 27 MEDLINE on STN
ACCESSION NUMBER: 2003273417 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12798493
TITLE: 1alpha,25(OH)2D3 causes a rapid increase in phosphatidylinositol-specific PLC-beta activity via phospholipase A2-dependent production of lysophospholipid.
AUTHOR: Schwartz Z; Shaked D; Hardin R R; Gruwell S; Dean D D; Sylvia V L; Boyan B D
CORPORATE SOURCE: Department of Biomedical Engineering, Georgia Institute of Technology, 315 Ferst Drive, Atlanta, GA 30332, USA.
CONTRACT NUMBER: DE-05937 (United States NIDCR)
DE-08603 (United States NIDCR)
SOURCE: Steroids, (2003 May) Vol. 68, No. 5, pp. 423-37.
Journal code: 0404536. ISSN: 0039-128X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200402
ENTRY DATE: Entered STN: 12 Jun 2003
Last Updated on STN: 2 Mar 2004
Entered Medline: 26 Feb 2004

AB 1alpha,25(OH)(2)D(3) activates protein kinase C (PKC) in rat growth plate chondrocytes via mechanisms involving phosphatidylinositol-specific phospholipase C (PI-PLC) and phospholipase A(2) (PLA(2)). The purpose of this study was to determine if 1alpha,25(OH)(2)D(3) activates PI-PLC directly or through a PLA(2)-dependent mechanism. We determined which PLC isoforms are present in the growth plate chondrocytes, and determined which isoform(s) of PLC is(are) regulated by 1alpha,25(OH)(2)D(3). Inhibitors and activators of PLA(2) were used to assess the inter-relationship between these two phospholipid-signaling pathways. PI-PLC activity in lysates of prehypertrophic and upper hypertrophic zone (growth zone) cells that were incubated with 1alpha,25(OH)(2)D(3), was increased within 30s with peak activity at 1-3 min. PI-PLC activity in resting zone cells was unaffected by 1alpha,25(OH)(2)D(3). 1beta,25(OH)(2)D(3), 24R,25(OH)(2)D(3), actinomycin D and cycloheximide had no effect on PLC in lysates of growth zone cells. Thus, 1alpha,25(OH)(2)D(3) regulation of PI-PLC enzyme activity is stereospecific, cell maturation-dependent, and nongenomic. PLA(2)-activation (mastoparan or melittin) increased PI-PLC activity to the same extent as 1alpha,25(OH)(2)D(3); PLA(2)-inhibition (quinacrine,

oleyloxyethylphosphorylcholine (OEPC), or AACOCF(3)) reduced the effect of 1alpha,25(OH)(2)D(3). Neither arachidonic acid (AA) nor its metabolites affected PI-PLC. In contrast, lysophosphatidylcholine (LPC) and lysophosphatidylethanolamine (LPE) activated PI-PLC (LPE>LPC). 1alpha,25(OH)(2)D(3) stimulated PI-PLC and PKC activities via Gq; GDPbetaS inhibited activity, but pertussis toxin did not. RT-PCR showed that the cells express PLC-beta1a, PLC-beta1b, PLC-beta3 and PLC-gamma1 mRNA. Antibodies to PLC-beta1 and PLC-beta3 blocked the 1alpha,25(OH)(2)D(3) effect; antibodies to PLC-delta and PLC-gamma did not. Thus, 1alpha,25(OH)(2)D(3) regulates PLC-beta through PLA(2)-dependent production of lysophospholipid.

L2 ANSWER 4 OF 27 MEDLINE on STN
ACCESSION NUMBER: 2003052135 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12562098
TITLE: Enzymatic activity and inhibition of the neurotoxic complex vipoxin from the venom of Vipera ammodytes meridionalis.
AUTHOR: Noetzel Corinna; Chandra Vikas; Perbandt Markus; Rajashankar Kanagalaghatta; Singh Tej; Aleksiev Boris; Kalkura Narayana; Genov Nicolay; Betzel Christian
CORPORATE SOURCE: Institute of Medical Biochemistry and Molecular Biology, University Hospital Eppendorf c/o DESY, Build. 22a, Notkestrasse 85, 22603 Hamburg, Germany.
SOURCE: Zeitschrift fur Naturforschung. C, Journal of biosciences, (2002 Nov-Dec) Vol. 57, No. 11-12, pp. 1078-83. Journal code: 8912155. ISSN: 0341-0382.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200304
ENTRY DATE: Entered STN: 4 Feb 2003
Last Updated on STN: 16 Apr 2003
Entered Medline: 14 Apr 2003
AB Vipoxin from the venom of Vipera ammodytes meridionalis is an unique neurotoxic complex between a toxic phospholipase A2 and a highly homologous non-toxic protein inhibitor. It is an example of evolution of a catalytic and toxic function into inhibitory and non-toxic one. The activity of the V. ammodytes meridionalis toxin is 1.7 times higher than that of the closely related (92% sequence identity) neurotoxic complex RV4/RV7 from the venom of Vipera russelli formosensis The enhanced enzymatic activity of vipoxin is attributed to limited structural changes, in particular to the substitutions G54R and Q78K in the PLA2 subunit of the complex and to the T54R substitution in the inhibitor. Oleyloxyethylphosphocholine, aristolochic acid and vitamin E suppressed the enzymatic activity of vipoxin and its isolated PLA2 subunit. These compounds influence inflammatory processes in which PLA2 is implicated. The peptide Lys-Ala-Ile-Tyr-Ser, which is an integral part of the PLA2 components of the two neurotoxic complexes from V. ammodytes meridionalis and V. russelli formosensis (sequence 70-74) activated vipoxin increasing its PLA2 activity by 23%. This is in contrast to the inhibitory effect of the respective pentapeptides with 70-74 sequences on other group II PLA2s. Surprisingly, the same peptide inhibited 46% of the V. russelli formosensis PLA2 activity. The limited changes in the structure of the two highly homologous neurotoxins lead to considerable differences in their interaction with native peptides.

L2 ANSWER 5 OF 27 MEDLINE on STN
ACCESSION NUMBER: 2002725402 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12489129
TITLE: Eicosanoids in insect immunity: bacterial infection

stimulates hemocytic phospholipase A2 activity in tobacco hornworms.
AUTHOR: Tunaz Hasan; Park Youngjin; Buyukguzel Kemal; Bedick Jon C; Nor Aliza A R; Stanley David W
CORPORATE SOURCE: Insect Biochemical Physiology Laboratory, University of Nebraska, Lincoln 68583-0816, USA.
SOURCE: Archives of insect biochemistry and physiology, (2003 Jan) Vol. 52, No. 1, pp. 1-6.
Journal code: 8501752. ISSN: 0739-4462.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200303
ENTRY DATE: Entered STN: 19 Dec 2002
Last Updated on STN: 4 Mar 2003
Entered Medline: 3 Mar 2003

AB Intracellular phospholipase A(2) (PLA(2)) is responsible for releasing arachidonic acid from cellular phospholipids, and is thought to be the first step in eicosanoid biosynthesis. Intracellular PLA(2)s have been characterized in fat body and hemocytes from tobacco hornworms, Manduca sexta. Here we show that bacterial challenge stimulated increased PLA(2) activity in isolated hemocyte preparations, relative to control hemocyte preparations that were challenged with water. The increased activity was detected as early as 15 s post-challenge and lasted for at least 1 h. The increased activity depended on a minimum bacterial challenge dose, and was inhibited in reactions conducted in the presence of oleyoxyethylphosphorylcholine, a site-specific PLA(2) inhibitor. In independent experiments with serum prepared from whole hemolymph, we found no PLA(2) activity was secreted into serum during the first 24 h following bacterial infection. We infer that a hemocytic intracellular PLA(2) activity is increased immediately an infection is detected. The significance of this enzyme lies in its role in launching the biosynthesis of eicosanoids, which mediate cellular immune reactions to bacterial infection.

L2 ANSWER 6 OF 27 MEDLINE on STN
ACCESSION NUMBER: 2002147899 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11841807
TITLE: Identification of the phospholipase A(2) isoforms that contribute to arachidonic acid release in hypoxic endothelial cells: limits of phospholipase A(2) inhibitors.
AUTHOR: Michiels Carine; Renard Patricia; Bouaziz Najat; Heck Nathalie; Eliaers Francois; Ninane Noelle; Quarck Rozenn; Holvoet Paul; Raes Martine
CORPORATE SOURCE: Laboratoire de Biochimie et Biologie Cellulaire, Facultes Universitaires Notre Dame de la Paix, 61 rue de Bruxelles, 5000, Namur, Belgium.. carine.michiels@fundp.ac.be
SOURCE: Biochemical pharmacology, (2002 Jan 15) Vol. 63, No. 2, pp. 321-32.
Journal code: 0101032. ISSN: 0006-2952.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200203
ENTRY DATE: Entered STN: 8 Mar 2002
Last Updated on STN: 3 Apr 2002
Entered Medline: 27 Mar 2002

AB Changes in endothelium functions during ischemia are thought to be of importance in numerous pathological conditions, with, for instance, an increase in the release of inflammatory mediators like prostaglandins. Here, we showed that hypoxia increases phospholipase A(2) (PLA(2)) activity in human umbilical vein endothelial cells. Both basal PLA(2) activity and PG synthesis are sensitive to BEL and AACOCF3, respectively, inhibitors of calcium-independent PLA(2) (iPLA(2)) and cytosolic PLA(2) (cPLA(2)), while OPC, an inhibitor of soluble PLA(2) (sPLA(2)) only inhibited the hypoxia-induced AA release and PGF(2alpha) synthesis. Hypoxia does not alter expression of iPLA(2), sPLA(2) and cPLA(2) and cycloheximide did not inhibit PLA(2) activation, indicating that hypoxia-induced increase in PLA(2) activity is due to activation rather than induction. However, mRNA levels for sPLA(2) displayed a 2-fold increase after 2 hr incubation under hypoxia. BAPTA, an intracellular calcium chelator, partially inhibited the AA release in normoxia and in hypoxia. Direct assays of specific PLA(2) activity showed an increase in sPLA(2) activity but not in cPLA(2) activity after 2hr hypoxia. Taken together, these results indicate that the hypoxia-induced increase in PLA(2) activity is mostly due to the activation of sPLA(2).

L2 ANSWER 7 OF 27 MEDLINE on STN
ACCESSION NUMBER: 2001301904 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11226404
TITLE: The involvement of phospholipase A(2) in ethanol-induced gastric muscle contraction.
AUTHOR: Sim S S; Choi J C; Min D S; Rhie D J; Yoon S H; Hahn S J; Kim C J; Kim M S; Jo Y H
CORPORATE SOURCE: Department of Pathophysiology, College of Pharmacy, Chung-Ang University, 221 Huksuk-dong, Dongjak-gu, Seoul 156-756, South Korea.
SOURCE: European journal of pharmacology, (2001 Feb 16) Vol. 413, No. 2-3, pp. 281-5.
Journal code: 1254354. ISSN: 0014-2999.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200105
ENTRY DATE: Entered STN: 4 Jun 2001
Last Updated on STN: 4 Jun 2001
Entered Medline: 31 May 2001
AB To understand the underlying mechanism of ethanol in tonic contraction, the effect of ethanol on phospholipase A(2) and phospholipase C activities and the effects of phospholipase inhibitors on ethanol-induced contraction of cat gastric smooth muscle were tested. Circular muscle strips (2.0 x 0.2 cm) obtained from the fundus of cat stomach were used to measure isometric contraction. Ethanol elicited tonic contraction and activated phospholipase A(2) activity in a dose-dependent manner. Phospholipase A(2) inhibitors, manoalide (0.1--10 microM) and oleyloxyethyl phosphorylcholine (1--10 microM), significantly inhibited ethanol-induced contraction. Furthermore, 342 mM ethanol-induced contraction was significantly inhibited by cyclooxygenase inhibitors, ibuprofen (10--100 microM) and indomethacin (10--100 microM), but not by lipoxygenase inhibitors. On the other hand, phospholipase C inhibitors had no effect on ethanol-induced contraction, indicating that phospholipase C is not involved in ethanol-induced contraction. It is suggested from the above results that ethanol-induced contraction in cat gastric smooth muscle is, in part, mediated by phospholipase A(2) and cyclooxygenase pathways.

L2 ANSWER 8 OF 27 MEDLINE on STN
ACCESSION NUMBER: 2001121483 MEDLINE

DOCUMENT NUMBER: PubMed ID: 10991918
TITLE: Investigation into the involvement of phospholipases A(2) and MAP kinases in modulation of AA release and cell growth in A549 cells.
AUTHOR: Choudhury Q G; McKay D T; Flower R J; Croxtall J D
CORPORATE SOURCE: Department of Biochemical Pharmacology, The William Harvey Research Institute, St. Bartholomew's and the Royal London School of Medicine and Dentistry (Queen Mary and Westfield College), Charterhouse Square, London EC1M 6BQ.
SOURCE: British journal of pharmacology, (2000 Sep) Vol. 131, No. 2, pp. 255-65.
Journal code: 7502536. ISSN: 0007-1188.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200102
ENTRY DATE: Entered STN: 22 Mar 2001
Last Updated on STN: 22 Mar 2001
Entered Medline: 22 Feb 2001

AB 1. We have investigated the contribution of specific PLA(2)s to eicosanoid release from A549 cells by using specific inhibitors of secretory PLA(2) (ONO-RS-82 and oleyloxyethylphosphocholine), cytosolic PLA(2) (AACOCF(3) and MAFP) and calcium-independent PLA(2) (HELSS, MAFP and PACOCF(3)). Similarly, by using specific inhibitors of p38 MAPK (SB 203580), ERK1/2 MAPK (Apigenin) and MEK1/2 (PD 98059) we have further evaluated potential pathways of AA release in this cell line. 2. ONO-RS-82 and oleyloxyethylphosphocholine had no significant effect on EGF or IL-1beta stimulated (3)H-AA or PGE(2) release or cell proliferation. AACOCF(3), HELSS, MAFP and PACOCF(3) significantly inhibited both EGF and IL-1beta stimulated (3)H-AA and PGE(2) release as well as cell proliferation. Apigenin and PD 98509 significantly inhibited both EGF and IL-1beta stimulated (3)H-AA and PGE(2) release and cell proliferation whereas, SB 203580 had no significant effect on EGF or IL-1beta stimulated (3)H-AA release, or cell proliferation but significantly suppressed EGF or IL-1beta stimulated PGE(2) release. 3. These results confirm that the liberation of AA release, generation of PGE(2) and cell proliferation is mediated largely through the actions of cPLA(2) whereas, sPLA(2) plays no significant role. We now also report a hitherto unsuspected contribution of iPLA(2) to this process and demonstrate that the stimulating action of EGF and IL-1beta in AA release and cell proliferation is mediated in part via a MEK and ERK-dependent pathway (but not through p38MAPK). We therefore propose that selective inhibitors of MEK and MAPK pathways may be useful in controlling AA release, eicosanoid production and cell proliferation.

L2 ANSWER 9 OF 27 MEDLINE on STN
ACCESSION NUMBER: 2000106581 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10643787
TITLE: Novel strategies for opposing murine microglial activation.
AUTHOR: Paris D; Town T; Mullan M
CORPORATE SOURCE: The Roskamp Institute, University of South Florida, Tampa 33613, USA.. dparis@com1.med.usf.edu
SOURCE: Neuroscience letters, (2000 Jan 7) Vol. 278, No. 1-2, pp. 5-8.
Journal code: 7600130. ISSN: 0304-3940.
PUB. COUNTRY: Ireland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200002

ENTRY DATE: Entered STN: 9 Mar 2000
Last Updated on STN: 9 Mar 2000
Entered Medline: 22 Feb 2000

AB Pathologic microglial activation is believed to contribute to progressive neuronal damage in neurodegenerative diseases by the release of potentially neurotoxic agents, such as pro-inflammatory cytokines including tumor necrosis factor alpha (TNF-alpha). Using cultured N9 microglial cells, we have examined the regulation of TNF-alpha following endotoxic insult with lipopolysacharide (LPS), focusing on the role of the pro-inflammatory phospholipase A2/mitogen activated protein kinase/arachidonic acid/cyclo-oxygenase-2 cascade and the nitric oxide/cGMP pathway. Data show that various inhibitors of the PLA2 cascade markedly inhibit LPS-induced TNF-alpha release, supporting a key role of this pathway in the regulation of microglial activation. We also investigated the putative effects of cGMP-elevating agents on blocking microglial activation induced by LPS. Data show that each member of this class of cGMP-elevating compounds that we employed opposed microglial TNF-alpha release, suggesting that strengthening intracellular cGMP signaling mitigates against microglial activation. Taken together, our results suggest novel strategies for reducing microglial activation.

L2 ANSWER 10 OF 27 MEDLINE on STN
ACCESSION NUMBER: 1999318341 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10391457
TITLE: Involvement of axonal phospholipase A2 activity in the outgrowth of adult mouse sensory axons in vitro.
AUTHOR: Hornfelt M; Ekstrom P A; Edstrom A
CORPORATE SOURCE: Department of Animal Physiology, Lund University, Sweden.
SOURCE: Neuroscience, (1999) Vol. 91, No. 4, pp. 1539-47.
Journal code: 7605074. ISSN: 0306-4522.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199908
ENTRY DATE: Entered STN: 10 Sep 1999
Last Updated on STN: 10 Sep 1999
Entered Medline: 24 Aug 1999

AB The effect on axonal outgrowth of inhibition of phospholipase A2 activity was studied in a recently developed in vitro model, where dorsal root ganglia with attached spinal roots and nerve stumps from young adult mice were cultured in an extracellular matrix material (Matrigel). The phospholipase A2 inhibitors 4-bromophenacyl bromide and oleyloxyethyl phosphorylcholine dose-dependently reduced axonal outgrowth from the sciatic nerve stump. A similar inhibitory effect was seen when only the cut nerve end was exposed to the inhibitors in a compartmental culture system. The local effect of phospholipase A2 inhibition was further investigated on axons established in culture, using time-lapse recording. Exposure to phospholipase A2 inhibitors caused the retraction of filopodia extensions and a reduction in growth cone motility within a few minutes. After removal of inhibition, normal growth cone motility and axonal growth were regained. Nerve cell bodies and axons, in contrast to Schwann cells, showed immunoreactivity after staining with an antiserum against secretory phospholipase A2, and elevated levels of the enzyme could be detected after culture for 24 h. The immunoreactive protein was of approximately 170,000 molecular weight (phospholipase A2-170) as determined by sodium dodecyl sulphate-polyacrylamide gel electrophoresis and immunoblotting. The localization of phospholipase A2-170 in axons growing into the Matrigel was also demonstrated by use of a whole-mount technique. The results of this study show the importance of continuous phospholipase A2 activity for growth cone motility and axonal outgrowth in the mammalian

peripheral nerve, and suggest the involvement of an axonally localized enzyme.

L2 ANSWER 11 OF 27 MEDLINE on STN
ACCESSION NUMBER: 1999217849 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10203186
TITLE: Role of endothelial factors in the specific response of mouse tumour-feeding arterioles to stimulation of 5-HT1 receptors.
AUTHOR: Laemmel E; Stucker O; Vicaut E
CORPORATE SOURCE: Dept de Biophysique et INSERM U141, Hopital F. Widal, Paris, France.
SOURCE: International journal of radiation biology, (1999 Mar) Vol. 75, No. 3, pp. 365-71.
Journal code: 8809243. ISSN: 0955-3002.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Space Life Sciences
ENTRY MONTH: 199904
ENTRY DATE: Entered STN: 11 May 1999
Last Updated on STN: 11 May 1999
Entered Medline: 29 Apr 1999
AB PURPOSE: To investigate the possible role of endothelial mediators on the increased vasoconstriction to 5-HT1 receptor stimulation by the host-modified arterioles feeding a Meth-A tumour implanted in the flank of female Balb/c mice. MATERIALS AND METHODS: Using intravital microscopy, the response to the topical administration of the general 5-HT1 agonist 5-carboxamidotryptamine maleate (5-CT; 10(-6) M to 10(-4) M) by the tumour-feeding arterioles with the responses of tumour-independent arterioles and those of control arterioles from mice without tumour after antagonization or inhibition of the synthesis of endothelial mediators was compared. RESULTS: The dramatically higher vasoconstriction to 5-CT observed in tumour-feeding arterioles than in tumour-independent or control arterioles still persisted when either nitric oxide synthase, cyclooxygenase, lipoxygenase, or phospholipase A2 were inhibited or when thromboxane A2 or endothelin were antagonized. CONCLUSIONS: It was concluded that the higher reactivity to 5-HT1 stimulation by tumour-feeding arterioles is not due to changes in endothelial mediator release but probably due to changes affecting arteriolar smooth muscle.

L2 ANSWER 12 OF 27 MEDLINE on STN
ACCESSION NUMBER: 1998363084 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9699504
TITLE: Arachidonic acid is an autocoid mediator of the differential action of 1,25-(OH)2D3 and 24,25-(OH)2D3 on growth plate chondrocytes.
AUTHOR: Boyan B D; Sylvia V L; Curry D; Chang Z; Dean D D; Schwartz Z
CORPORATE SOURCE: Department of Orthopaedics, The University of Texas Health Science Center at San Antonio, 78284-7774, USA.. messier@uthscsa.edu
CONTRACT NUMBER: DE-05937 (United States NIDCR)
DE-08603 (United States NIDCR)
SOURCE: Journal of cellular physiology, (1998 Sep) Vol. 176, No. 3, pp. 516-24.
Journal code: 0050222. ISSN: 0021-9541.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 199808
ENTRY DATE: Entered STN: 3 Sep 1998
Last Updated on STN: 3 Mar 2000
Entered Medline: 21 Aug 1998

AB Prior studies have shown that 24,25-(OH)2D3 and 1,25-(OH)2D3 regulate protein kinase C (PKC) in costochondral chondrocytes in a cell maturation-dependent manner, with 1,25-(OH)2D3 affecting primarily growth zone (GC) cells and 24,25-(OH)2D3 affecting primarily resting zone (RC) cells. In addition, 1,25-(OH)2D3 has been shown to increase phospholipase A2 activity in GC, while 24,25-(OH)2D3 has been shown to decrease phospholipase A2 activity in RC. Stimulation of phospholipase A2 in GC caused an increase in PKC, whereas inhibition of phospholipase A2 activity in RC cultures increased both basal and 24,25-(OH)2D3-induced PKC activity, suggesting that phospholipase A2 may play a central role in mediating the effects of the vitamin D metabolites on PKC. To test this hypothesis, RC and GC cells were cultured in the presence and absence of phospholipase A2 inhibitors (quinacrine and oleyloxyethylphosphorylcholine [OEPC]), phospholipase A2 activators (melittin and mastoparan), or arachidonic acid alone or in the presence of the target cell-specific vitamin D metabolite. PKC specific activity in the cell layer was determined as a function of time. Phospholipase A2 inhibitors decreased both basal and 1,25-(OH)2D3-induced PKC activity in GC. When phospholipase A2 activity was activated by inclusion of melittin or mastoparan in the cultures, basal PKC activity in RC was reduced, while that in GC was increased. Similarly, melittin and mastoparan decreased 24,25-(OH)2D3-induced PKC activity in RC and increased 1,25-(OH)2D3-induced PKC activity in GC. For both cell types, the addition of arachidonic acid to the culture media produced an effect on PKC activity that was similar to that observed when phospholipase A2 activators were added to the cells. These results demonstrate that vitamin D metabolite-induced changes in phospholipase A2 activity are directly related to changes in PKC activity. Similarly, exogenous arachidonic acid affects PKC in a manner consistent with activation of phospholipase A2. These effects are cell maturation- and time-dependent and metabolite-specific.

L2 ANSWER 13 OF 27 MEDLINE on STN
ACCESSION NUMBER: 1991183640 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1901255
TITLE: Inhibitors of cytochrome P-450 attenuate the myogenic response of dog renal arcuate arteries.
AUTHOR: Kauser K; Clark J E; Masters B S; Ortiz de Montellano P R; Ma Y H; Harder D R; Roman R J
CORPORATE SOURCE: Department of Physiology, Medical College of Wisconsin, Milwaukee 53226.
CONTRACT NUMBER: HL-29587 (United States NHLBI)
HL-33833 (United States NHLBI)
HL-36279 (United States NHLBI)
+
SOURCE: Circulation research, (1991 Apr) Vol. 68, No. 4, pp. 1154-63.
Journal code: 0047103. ISSN: 0009-7330.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199105
ENTRY DATE: Entered STN: 26 May 1991
Last Updated on STN: 3 Feb 1997

Entered Medline: 8 May 1991

AB The role of cytochrome P-450 in the myogenic response of isolated, perfused renal arcuate arteries of dogs to elevations in transmural pressure was examined. The phospholipase A2 inhibitor oleyloxyethylphosphorylcholine (1 and 10 microM) inhibited the greater than threefold increase in active wall tension in these arteries after an elevation in perfusion pressure from 80 to 160 mm Hg. Inhibition of cyclooxygenase activity with indomethacin (1 or 10 microM) had no effect on this response. The cytochrome P-450 inhibitors ketoconazole (10 and 100 microM) and beta-diethyl-aminoethyldiphenylpropylacetate (SKF 525A, 10 and 100 microM) also inhibited the myogenic response. At a pressure of 160 mm Hg, SKF 525A (10 microM) and ketoconazole (100 microM) reduced active wall tension in renal arteries by approximately 70%. Partial inhibition of the myogenic response was obtained after perfusion of the vessels with mechanism-based inhibitors of P-450, 1-aminobenzotriazole (75 microM) and 12-hydroxy-16-heptadecynoic acid (20 microM). The thromboxane receptor antagonist SQ 29,548 (1 or 10 microM) had no effect on the pressure-induced increase in active wall tension in renal arteries. Arachidonic acid (50 microM) constricted isolated perfused renal arteries and potentiated the myogenic response in the presence of indomethacin. This response was completely reversed by ketoconazole (100 microM) or SKF 525A (100 microM). Microsomes (1 mg/ml) prepared from small renal arteries (200-500 microns) and incubated with [1-14C]arachidonic acid (0.5 mu Ci, 50 microM) produced a metabolite that coeluted with 20-hydroxyeicosatetraenoic acid (20-HETE) during reversed-phase high-performance liquid chromatography. The formation of this product was inhibited by both ketoconazole and SKF 525A at concentrations of 10 and 100 microM. These results are consistent with the involvement of the vasoconstrictor 20-HETE and other cytochrome P-450 metabolites of endogenous fatty acids in the myogenic response.

L2 ANSWER 14 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2004:829238 CAPLUS

DOCUMENT NUMBER: 141:329077

TITLE: Interactions of 12-lipoxygenase with phospholipase A2 isoforms following platelet activation through the glycoprotein VI collagen receptor

AUTHOR(S): Coffey, Marcus J.; Coles, Barbara; Locke, Matthew; Bermudez-Fajardo, Alexandra; Williams, P. Claire; Jarvis, Gavin E.; O'Donnell, Valerie B.

CORPORATE SOURCE: Department of Medical Biochemistry and Immunology, Wales College of Medicine, Cardiff University, Cardiff, CF14 4XN, UK

SOURCE: FEBS Lett. (2004), 576(1-2), 165-168
CODEN: FEBLAL; ISSN: 0014-5793

PUBLISHER: Elsevier B.V.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Recent studies implicate the collagen receptor, glycoprotein VI (GPVI) in activation of platelet 12-lipoxygenase (p12-LOX). Herein, we show that GPVI-stimulated 12-hydro(peroxy)eicosatetraenoic acid (H(P)ETE) synthesis is inhibited by palmityl trifluromethyl ketone or oleyloxyethylphosphocholine, but not bromoenol lactone, implicating secretory and cytosolic, but not calcium-independent phospholipase A2 (PLA2) isoforms. Also, following GPVI activation, 12-LOX co-immunoppts. with both cytosolic and secretory PLA2 (sPLA2). Finally, venoms containing sPLA2 acutely activate p12-LOX in a dose-dependent manner. This study shows that platelet 12-H(P)ETE generation utilizes arachidonate substrate from both c- and sPLA2 and that 12-LOX functionally assocs. with both PLA2 isoforms.

REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 15 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2004:748810 CAPLUS
DOCUMENT NUMBER: 142:129558
TITLE: Phospholipase A2 in salivary glands isolated from tobacco hornworms, Manduca sexta
AUTHOR(S): Tunaz, Hasan; Stanley, David W.
CORPORATE SOURCE: Insect Biochemical Physiology Laboratory, University of Nebraska-Lincoln, Lincoln, NE, 68583-0816, USA
SOURCE: Comparative Biochemistry and Physiology, Part B: Biochemistry & Molecular Biology (2004), 139B(1), 27-33
CODEN: CBPBB8; ISSN: 1096-4959
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A phospholipase A2 (PLA2) associated with the salivary glands of M. sexta is described. This enzyme was able to hydrolyze arachidonic acid from the sn-2 position of phospholipases. The addition of the Ca2+-chelator, EGTA, or Ca2+, to the Tris reaction buffer impaired the PLA2 activity, from which it was inferred that the enzyme required very low concns. of Ca2+ or perhaps other ions for optimal activity. PLA2 activity was sensitive to protein concentration (highest activity at 25 μg protein/μL), reaction time (optimal at 30 min), buffer pH (optimal at pH 8-10), and reaction temperature (optimal range 18-38°). The salivary gland PLA2 was sensitive to the site-specific inhibitor, oleyloxyethylphosphorylcholine and stable to freezing at -80°, but not -20°. The biol. significance of this enzyme may relate to hydrolysis of fatty acid moieties from dietary phospholipases for absorption by midgut epithelia. This salivary gland enzyme may also be responsible for killing food-borne bacteria.
REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 16 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2003:447810 CAPLUS
DOCUMENT NUMBER: 139:286724
TITLE: 1α,25(OH)2D3 causes a rapid increase in phosphatidylinositol-specific PLC-β activity via phospholipase A2-dependent production of lysophospholipid
AUTHOR(S): Schwartz, Z.; Shaked, D.; Hardin, R. R.; Gruwell, S.; Dean, D. D.; Sylvia, V. L.; Boyan, B. D.
CORPORATE SOURCE: Department of Biomedical Engineering, Georgia Institute of Technology, Atlanta, GA, 30332, USA
SOURCE: Steroids (2003), 68(5), 423-437
CODEN: STEDAM; ISSN: 0039-128X
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB 1α,25(OH)2D3 activates protein kinase C (PKC) in rat growth plate chondrocytes via mechanisms involving phosphatidylinositol-specific phospholipase C (PI-PLC) and phospholipase A2 (PLA2). The purpose of this study was to determine if 1α,25(OH)2D3 activates PI-PLC directly or through a PLA2-dependent mechanism. We determined which PLC isoforms are present in the growth plate chondrocytes, and determined which isoform(s) of PLC is(are) regulated by 1α,25(OH)2D3. Inhibitors and activators of PLA2 were used to assess the inter-relationship between these two phospholipid-signaling pathways. PI-PLC activity in lysates of pre-hypertrophic and upper hypertrophic zone (growth zone) cells that were incubated with 1α,25(OH)2D3, was increased within 30 s with peak activity at 1-3 min. PI-PLC activity in resting zone cells was unaffected

by 1α,25(OH)2D3. 1β,25(OH)2D3, 24R,25(OH)2D3, actinomycin D and cycloheximide had no effect on PLC in lysates of growth zone cells. Thus, 1α,25(OH)2D3 regulation of PI-PLC enzyme activity is stereospecific, cell maturation-dependent, and nongenomic. PLA2-activation (mastoparan or melittin) increased PI-PLC activity to the same extent as 1α,25(OH)2D3; PLA2-inhibition (quinacrine, oleyloxyethylphosphorylcholine (OEPC), or AACOCF3) reduced the effect of 1α,25(OH)2D3. Neither arachidonic acid (AA) nor its metabolites affected PI-PLC. In contrast, lysophosphatidylcholine (LPC) and lysophosphatidylethanolamine (LPE) activated PI-PLC (LPE>LPC). 1α,25(OH)2D3 stimulated PI-PLC and PKC activities via Gq; GDPβS inhibited activity, but pertussis toxin did not. RT-PCR showed that the cells express PLC-β1a, PLC-β1b, PLC-β3 and PLC-γ1 mRNA. Antibodies to PLC-β1 and PLC-β3 blocked the 1α,25(OH)2D3 effect; antibodies to PLC-δ and PLC-γ did not. Thus, 1α,25(OH)2D3 regulates PLC-β through PLA2-dependent production of lysophospholipid.

REFERENCE COUNT: 80 THERE ARE 80 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 17 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2003:112769 CAPLUS

DOCUMENT NUMBER: 139:346941

TITLE: Enzymatic activity and inhibition of the neurotoxic complex vipoxin from the venom of Vipera ammodytes meridionalis

AUTHOR(S): Noetzel, Corinna; Chandra, Vikas; Perbandt, Markus; Rajashankar, Kanagalaghatta; Singh, Tej; Aleksiev, Boris; Kalkura, Narayana; Genov, Nicolay; Betzel, Christian

CORPORATE SOURCE: Institute of Medical Biochemistry and Molecular Biology, University Hospital Eppendorf, Hamburg, 22603, Germany

SOURCE: Zeitschrift fuer Naturforschung, C: Journal of Biosciences (2002), 57(11/12), 1078-1083
CODEN: ZNCBDA; ISSN: 0939-5075

PUBLISHER: Verlag der Zeitschrift fuer Naturforschung

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Vipoxin from the venom of Vipera ammodytes meridionalis is an unique neurotoxic complex between a toxic phospholipase A2 and a highly homologous non-toxic protein inhibitor. It is an example of evolution of a catalytic and toxic function into inhibitory and non-toxic one. The activity of the V. ammodytes meridionalis toxin is 1.7 times higher than that of the closely related (92% sequence identity) neurotoxic complex RV4/RV7 from the venom of Vipera russelli formosensis. The enhanced enzymic activity of vipoxin is attributed to limited structural changes, in particular to the substitutions G54R and Q78K in the PLA2 subunit of the complex and to the T54R substitution in the inhibitor. Oleyloxyethylphosphocholine, aristolochic acid and vitamin E suppressed the enzymic activity of vipoxin and its isolated PLA2 subunit. These compds. influence inflammatory processes in which PLA2 is implicated. The peptide Lys-Ala-Ile-Tyr-Ser, which is an integral part of the PLA2 components of the two neurotoxic complexes from V. ammodytes meridionalis and V. russelli formosensis (sequence 70-74) activated vipoxin increasing its PLA2 activity by 23%. This is in contrast to the inhibitory effect of the resp. pentapeptides with 70-74 sequences on other group II PLA2s. Surprisingly, the same peptide inhibited 46% of the V. russelli formosensis PLA2 activity. The limited changes in the structure of the two highly homologous neurotoxins lead to considerable differences in their interaction with native peptides.

REFERENCE COUNT: 19 THERE ARE 19 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 18 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2000:735786 CAPLUS
DOCUMENT NUMBER: 133:345041
TITLE: Investigation into the involvement of phospholipases A2 and MAP kinases in modulation of AA release and cell growth in A549 cells
AUTHOR(S): Choudhury, Qamrul G.; Mckay, Diane T.; Flower, Roderick J.; Croxtall, Jamie D.
CORPORATE SOURCE: Department of Biochemical Pharmacology, The William Harvey Research Institute, St. Bartholomew's and the Royal London School of Medicine and Dentistry (Queen Mary and Westfield College, London, EC1M 6BQ, UK
SOURCE: British Journal of Pharmacology (2000), 131(2), 255-265
CODEN: BJPCBM; ISSN: 0007-1188
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The authors have investigated the contribution of specific PLA2s to eicosanoid release from A549 cells by using specific inhibitors of secretory PLA2 (ONO-RS-82 and oleyloxyethylphosphocholine), cytosolic PLA2 (AACOCF3 and MAFP) and calcium-independent PLA2 (HELSS, MAFP and PACOCF3). Similarly, by using specific inhibitors of p38 MAPK (SB 203580), ERK1/2 MAPK (Apigenin) and MEK1/2 (PD 98059) the authors have further evaluated potential pathways of AA release in this cell line. ONO-RS-82 and oleyloxyethylphosphocholine had no significant effect on EGF or IL-1β stimulated 3H-AA or PGE2 release or cell proliferation. AACOCF3, HELSS, MAFP and PACOCF3 significantly inhibited both EGF and IL-1β stimulated 3H-AA and PGE2 release as well as cell proliferation. Apigenin and PD 98509 significantly inhibited both EGF and IL-1β stimulated 3H-AA and PGE2 release and cell proliferation, whereas, SB 203580 had no significant effect on EGF or IL-1β stimulated 3H-AA release, or cell proliferation but significantly suppressed EGF or IL-1β stimulated PGE2 release. These results confirm that the liberation of AA release, generation of PGE2 and cell proliferation is mediated largely through the actions of cPLA2 whereas, sPLA2 plays no significant role. The authors now also report a hitherto unsuspected contribution of iPLA2 to this process and demonstrate that the stimulating action of EGF and IL-1β in AA release and cell proliferation is mediated in part via a MEK and ERK-dependent pathway (but not through p38MAPK). The authors therefore propose that selective inhibitors of MEK and MAPK pathways may be useful in controlling AA release, eicosanoid production and cell proliferation.
REFERENCE COUNT: 65 THERE ARE 65 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 19 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1999:641140 CAPLUS
DOCUMENT NUMBER: 132:21206
TITLE: Tissue polyunsaturated fatty acids and a digestive phospholipase A2 in the primary screwworm, Cochliomyia hominivorax
AUTHOR(S): Nor Aliza, A. R.; Rana, R. L.; Skoda, S. R.; Berkebile, D. R.; Stanley, D. W.
CORPORATE SOURCE: Insect Biochemical Physiology Laboratory, University of Nebraska, Lincoln, NE, USA
SOURCE: Insect Biochemistry and Molecular Biology (1999), 29(11), 1029-1038
CODEN: IBMBES; ISSN: 0965-1748
PUBLISHER: Elsevier Science Ltd.

DOCUMENT TYPE: Journal
LANGUAGE: English
AB The authors report on the presence of arachidonic acid in larval and adult tissues of the primary screwworm, Cochliomyia hominivorax, and of the secondary screwworm, C. macellaria. Arachidonic acid is present in the phospholipids of whole animal exts. of both species. This fatty acid appears to be accumulated during the larval stages, because proportions of arachidonic acid were higher in adults than in larvae. These insects probably obtain the arachidonic acid from dietary phospholipids. The authors also report on a phospholipase A2 activity in midgut prepns. from third instars of the primary screwworm. Phospholipase A2 is responsible for hydrolyzing fatty acids from the sn-2 position of dietary phospholipids to release essential fatty acids. The screwworm enzyme is similar to mammalian digestive phospholipase A2 because it depends on calcium for high catalytic activity, it is sensitive to the site-specific inhibitor oleyloxyethylphosphorylcholine, and it interacts with heparin. The authors further characterized the screwworm midgut phospholipase A2 by altering the reaction conditions, including reaction time, radioactive substrate concentration, protein concentration, pH and temperature The authors speculate that the biol. significance of this enzyme relates to acquiring essential fatty acids, including arachidonic acid, from dietary phospholipids.
REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 20 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1998:626204 CAPLUS
DOCUMENT NUMBER: 129:328495
ORIGINAL REFERENCE NO.: 129:66942h,66943a
TITLE: A digestive phospholipase A2 in larval mosquitoes, Aedes aegypti
AUTHOR(S): Aliza, A. R. Nor; Stanley, David W.
CORPORATE SOURCE: Insect Biochemical Physiology Laboratory, Department of Entomology, University of Nebraska, Lincoln, NE, 68583-0816, USA
SOURCE: Insect Biochemistry and Molecular Biology (1998), 28(8), 561-569
CODEN: IBMBES; ISSN: 0965-1748
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB We report on the presence of phospholipase A2 activity in whole larva and midguts of the yellow fever mosquito A. aegypti. Phospholipase A2 is responsible for hydrolyzing fatty acids from the sn-2 position of dietary phospholipids to release essential fatty acids for normal larval and adult growth. In contrast to the mammalian digestive phospholipase A2 background, the A. aegypti phospholipase A2 was Ca2+ independent. We further characterized the mosquito midgut phospholipase A2 by altering the reaction conditions including incubation time, protein concns., pH, and temperature The site-specific PLA2 inhibitor oleyloxyethylphosphorylcholine failed to inhibit the enzyme at concns. <5000 μM. The phospholipase A2 activity was consistently high throughout the 4th instar, but fell to very low levels on the 1st day of pupation which is a non-feeding stage. The enzyme is regulated with respect to feeding activity because fasting and re-feeding modified the larval digestive PLA2 activity.
REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 21 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1998:503604 CAPLUS

DOCUMENT NUMBER: 129:226089
ORIGINAL REFERENCE NO.: 129:45841a,45844a
TITLE: Arachidonic acid is an autocoid mediator of the differential action of 1,25-(OH)2D3 and 24,25-(OH)2D3 on growth plate chondrocytes
AUTHOR(S): Boyan, B. D.; Sylvia, V. L.; Curry, D.; Chang, Z.; Dean, D. D.; Schwartz, Z.
CORPORATE SOURCE: Department of Orthopaedics, The University of Texas Health Science Center at San Antonio, San Antonio, TX, 78284-7774, USA
SOURCE: Journal of Cellular Physiology (1998), 176(3), 516-524
CODEN: JCLLAX; ISSN: 0021-9541
PUBLISHER: Wiley-Liss, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Prior studies have shown that 24,25-(OH)2D3 and 1,25-(OH)2D3 regulate protein kinase C (PKC) in costochondral chondrocytes in a cell maturation-dependent manner, with 1,25-(OH)2D3 affecting primarily growth zone (GC) cells and 24,25-(OH)2D3 affecting primarily resting zone (RC) cells. In addition, 1,25-(OH)2D3 has been shown to increase phospholipase A2 activity in GC, while 24,25-(OH)2D3 has been shown to decrease phospholipase A2 activity in RC. Stimulation of phospholipase A2 in GC caused an increase in PKC, whereas inhibition of phospholipase A2 activity in RC cultures increased both basal and 24,25-(OH)2D3-induced PKC activity, suggesting that phospholipase A2 may play a central role in mediating the effects of the vitamin D metabolites on PKC. To test this hypothesis, RC and GC cells were cultured in the presence and absence of phospholipase A2 inhibitors (quinacrine and oleyloxyethylphosphorylcholine [OEPC]), phospholipase A2 activators (melittin and mastoparan), or arachidonic acid alone or in the presence of the target cell-specific vitamin D metabolite. PKC specific activity in the cell layer was determined as a function of time. Phospholipase A2 inhibitors decreased both basal and 1,25-(OH)2D3-induced PKC activity in GC. When phospholipase A2 activity was activated by inclusion of melittin or mastoparan in the cultures, basal PKC activity in RC was reduced, while that in GC was increased. Similarly, melittin and mastoparan decreased 24,25-(OH)2D3-induced PKC activity in RC and increased 1,25-(OH)2D3-induced PKC activity in GC. For both cell types, the addition of arachidonic acid to the culture media produced an effect on PKC activity that was similar to that observed when phospholipase A2 activators were added to the cells. These results demonstrate that vitamin D metabolite-induced changes in phospholipase A2 activity are directly related to changes in PKC activity. Similarly, exogenous arachidonic acid affects PKC in a manner consistent with activation of phospholipase A2. These effects are cell maturation- and time-dependent and metabolite-specific.
REFERENCE COUNT: 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 22 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1998:481432 CAPLUS
DOCUMENT NUMBER: 129:186925
ORIGINAL REFERENCE NO.: 129:37913a,37916a
TITLE: A digestive phospholipase A2 in midguts of tobacco hornworms, Manduca sexta L.
AUTHOR(S): Rana, Rico L.; Sarath, Gautam; Stanley, David W.
CORPORATE SOURCE: Insect Biochemistry/Physiology Laboratory, Department of Entomology, University of Nebraska, Lincoln, NE, 68583-0816, USA
SOURCE: Journal of Insect Physiology (1998), 44(3-4), 297-303
CODEN: JIPHAF; ISSN: 0022-1910
PUBLISHER: Elsevier Science Ltd.

DOCUMENT TYPE: Journal
LANGUAGE: English
AB We hypothesized that phospholipase A2 (PLA2) is a common feature of insect digestive physiol. PLA2 hydrolyzes polyunsatd. fatty acids (PUFAs) associated with the sn-2 position of phospholipids (PLs). We describe here a PLA2 from midgut contents of the tobacco hornworm, M. sexta. The enzyme is sensitive to pH (inactivated at low pH), protein concentration (<1.6 μg/μl), substrate concentration (<1.4 nmol/reaction), temperature (<30°), and incubation time. PLA2 activity is higher in fed than in starved larvae, and enzyme activity is associated with the midgut contents, rather than the midgut epithelium of fed larvae. All known secretory PLA2s, except for a PLA2 in venom of the marine snail, Conus magus, require high calcium concns. for catalysis, but the Manduca PLA2 appears to be calcium independent, and it exhibits increased PLA2 activity in the presence of a calcium chelator, EGTA. In addition, the partially purified Manduca PLA2 is not inhibited by the phospholipid analog, oleyloxyethylphosphorylcholine. These findings suggest that the Manduca digestive PLA2 may represent another novel form of PLA2.
REFERENCE COUNT: 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

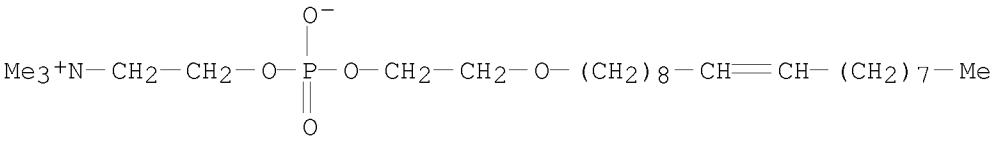
L2 ANSWER 23 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1993:188162 CAPLUS
DOCUMENT NUMBER: 118:188162
ORIGINAL REFERENCE NO.: 118:32247a,32250a
TITLE: The possible involvement of protein kinase C and phospholipase A2 in Hydra tentacle regeneration
AUTHOR(S): De Petrocellis, L.; Di Marzo, V.; Cimino, G.
CORPORATE SOURCE: Ist. Cibern., CNR, Arco Felice, I-80072, Italy
SOURCE: Experientia (1993), 49(1), 57-64
CODEN: EXPEAM; ISSN: 0014-4754
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The participation of protein kinase C (PKC) in the regeneration of tentacles of Hydra vulgaris was studied. Regeneration was induced by 1,2-sn-dioctanoyl-glycerol (diC8) and the novel diterpenoid diacylglycerol verrucosin B (VB), a potent PKC activator extracted from marine sources. VB substantially increased Hydra average tentacle number (ATN) at concns. 10,000 times lower than those needed for diC8 to exert an analogous effect. When both synthetic and natural VB analogs were tested, the structure/activity relation found in Hydra tentacle regeneration was identical to that known for DAG-induced activation of PKC in vitro. VB-induced increases in ATN was strongly counteracted by the PKC inhibitors sphingosine and A3, but was not synergistic with a 10-fold increase of extracellular Ca2+ concentration or with an increase of intracellular Ca2+ concentration obtained either with the ionophore A 23187 or with thapsigargin. This suggested the involvement of a non-Ca2+-dependent PKC in VB-triggered Hydra tentacle regeneration. The involvement of phospholipase A2 (PLA2) activation in Hydra regenerative processes was studied using the novel site-specific inhibitor of the enzyme, oleyloxyethylphosphorylcholine (OOPC), which brought about a striking inhibition of ATN in the low micromolar range. This effect was reversed by arachidonic acid (AA), while an enhancement of ATN was also observed with an inhibitor of AA uptake from membrane phospholipids, thus suggesting that PLA2-catalyzed liberation of AA is involved in Hydra tentacle regeneration. OOPC also blocked verrucosin B-induced PKC-mediated enhancement of ATN, thus suggesting that this effect is also mediated by PLA2 activation. ATN was increased also by compound 48/80, a direct activator of pertussis toxin-sensitive GTP-binding proteins, and this effect was counteracted by pertussis toxin pretreatment. None of the known AA cascade inhibitors exhibited an effect on ATN comparable to that exerted by OOPC, but, surprisingly, the cyclooxygenase inhibitor

indomethacin strongly enhanced ATN, thus suggesting that prostanoids might effect a neg. control on Hydra regenerative processes. This represents the 1st attempt to study the implication of >1 biochem. pathway as a signaling event in the hydroid regenerative processes.

L2 ANSWER 24 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1991:401629 CAPLUS
DOCUMENT NUMBER: 115:1629
ORIGINAL REFERENCE NO.: 115:342h,343a
TITLE: Inhibitors of cytochrome P-450 attenuate the myogenic response of dog renal arcuate arteries
AUTHOR(S): Kauser, Katalin; Clark, Joan E.; Masters, Bettie Sue; Ortiz de Montellano, Paul R.; Ma, Yunn Hwa; Harder, David R.; Roman, Richard J.
CORPORATE SOURCE: Dep. Physiol., Med. Coll. Wisconsin, Milwaukee, WI, 53226, USA
SOURCE: Circulation Research (1991), 68(4), 1154-63
CODEN: CIRUAL; ISSN: 0009-7330
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The role of cytochrome P 450 in the myogenic response of isolated, perfused renal arcuate arteries of dogs to elevations in transmural pressure was examined The phospholipase A2 inhibitor oleyloxyethylphosphorylcholine (1 and 10 μM) inhibited the 3-fold increase in active wall tension in these arteries after an elevation in perfusion pressure from 80 to 160 mm Hg. Inhibition of cyclooxygenase activity with indomethacin (1 or 10 μM) had no effect on this response. The cytochrome P 450 inhibitors ketoconazole (10 and 100 μM) and β-diethyl-aminoethyldiphenylpropylacetate (SKF 525A, 10 and 100 μM) also inhibited the myogenic response. At a pressure of 160 mm Hg, SKF 525A (10 μM) and ketoconazole (100 μM) reduced active wall tension in renal arteries by approx. 70%. Partial inhibition of the myogenic response was obtained after perfusion of the vessels with mechanism-based inhibitors of P 450, 1-aminobenzotriazole (75 μM) and 12-hydroxy-16-heptadecynoic acid (20 μM). The thromboxane receptor antagonist SQ 29,548 (1 of 10 μM) had no effect on the pressure-induced increase in active wall tension in renal arteries. Arachidonic acid (50 μM) constricted isolated perfused renal arteries and potentiated the myogenic response in the presence of indomethacin. This response was completely reversed by ketoconazole (100 μM) or SKF 525A (100 μM). Microsomes (1 mg/mL) prepared from small renal arteries (200-500 μm) and incubated with [1-14C]arachidonic acid (0.5 μCi, 50 μM) produced a metabolite that coeluted with 20-hydroxyeicosatetraenoic acid (20-HETE) during reversed-phase HPLC. The formation of this produce was inhibited by both ketoconazole and SKF 525A at concns. of 10 and 100 μM. These results are consistent with the involvement of the vasoconstrictor 20-HETE and other cytochrome P 450 metabolites of endogenous fatty acids in the myogenic response.

L2 ANSWER 25 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1985:406131 CAPLUS
DOCUMENT NUMBER: 103:6131
ORIGINAL REFERENCE NO.: 103:1103a,1106a
TITLE: A new efficient and versatile synthesis of alkyl phosphorylcholines
AUTHOR(S): Magolda, R. L.; Johnson, P. R.
CORPORATE SOURCE: Cent. Res. Dev. Dep., E. I. du Pont de Nemours and Co., Wilmington, DE, 19898, USA
SOURCE: Tetrahedron Letters (1985), 26(9), 1167-70
CODEN: TELEAY; ISSN: 0040-4039
DOCUMENT TYPE: Journal
LANGUAGE: English

OTHER SOURCE(S): CASREACT 103:6131
AB Phosphorylcholines ROP(O)(O-)OCH2CH2N+Me3 [R = Me(CH2)n, Me(CH2)7CH:CH(CH2)8, Me(CH2)mS(CH2)3, Me(CH2)7CH:CH(CH2)8S(CH2)3, Me(CH2)mOCH2CH2, Me(CH2)7CH:CH(CH2)8OCH2CH2; m = 15,17; r = 5,7,11,17] were prepared in 35-50% overall yield by treating ROH with POCl3, followed by ethylene glycol and treating the resulting cyclic phosphates with Me3N.
IT 96720-06-8P
RL: SPN (Synthetic preparation); PREP (Preparation)
(preparation of)
RN 96720-06-8 CAPLUS
CN 3,5,8-Trioxa-4-phosphahexacos-17-en-1-aminium, 4-hydroxy-N,N,N-trimethyl-, inner salt 4-oxide (CA INDEX NAME)



L2 ANSWER 26 OF 27 WPIDS COPYRIGHT 2008 THOMSON REUTERS on STN
ACCESSION NUMBER: 2000-171073 [15] WPIDS
DOC. NO. CPI: C2000-053194 [15]
TITLE: Modifying vasoactivity and inflammatory actions in microglia and neurons comprises regulating a soluble alphabeta pro-inflammatory pathway, useful for treating vascular diseases, e.g. cerebral amyloid angiopathy and vascular amyloidosis
DERWENT CLASS: B04
INVENTOR: MULLAN M J; PARIS D; TOWN T C
PATENT ASSIGNEE: (UYSF-N) UNIV SOUTH FLORIDA
COUNTRY COUNT: 84

PATENT INFO ABBR.:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC
WO 2000002561	A1	20000120	(200015)*	EN	77[29]	
AU 9949948	A	20000201	(200028)	EN		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000002561	A1	WO 1999-US15947	19990713
AU 9949948	A	AU 1999-49948	19990713

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9949948	A	Based on	WO 2000002561 A

PRIORITY APPLN. INFO: US 1998-92570P 19980713
AN 2000-171073 [15] WPIDS
AB WO 2000002561 A1 UPAB: 20060116
NOVELTY - Modifying vasoactivity comprises regulating a soluble alphabeta pro-inflammatory pathway.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:
(1) a pharmaceutical composition comprising an effective amount of

a soluble alphabeta pro-inflammatory pathway regulator and a carrier;

(2) a diagnostic method including detecting modification of the soluble alphabeta pro-inflammatory pathway; and

(3) a method of modifying inflammatory actions in microglia and neurons by regulating a soluble alphabeta pro-inflammatory pathway.

ACTIVITY - Vasoactive.

MECHANISM OF ACTION - Signal transduction pathway (e.g. sPLA2/MAPK/cPLA2/AA/LOX/COX) modulators.

In tests to evaluate the effect of alphabeta on microglial LTB4 release, soluble alphabeta treatment of the murine microglial cell line, N9, resulted in an increased release of LTB4. COX-2 inhibition (via the COX-2 specific inhibitor, NS-398, 50 microM) resulted in complete blockade of LTB4 release. Furthermore, inhibition of p38 MAPK by the specific inhibitor SB202190 (5 microM) or inhibition of MEK1/2 via PD98059 (25 microM) each resulted in complete blockade of alphabeta-induced microglial LTB4 release.

USE - The method is for treating patients with vascular disease by modifying an intracellular soluble alphabeta pro-inflammatory pathway (claimed). Vascular diseases to be treated include cerebral amyloid angiopathy and vascular amyloidosis.

ADVANTAGE - The methods reduce neuronal cell death associated with the pro-inflammatory pathway and vasoactivity.

L2 ANSWER 27 OF 27 USPATFULL on STN

ACCESSION NUMBER:	2006:127387 USPATFULL			
TITLE:	Coupling factor 6 inhibitor and potentiator and use thereof			
INVENTOR(S):	Osanai, Tomohiro, Nakatsugaru-gun, JAPAN Magota, Koji, Takatsuki, JAPAN			
PATENT ASSIGNEE(S):	Daiichi Suntory Pharma Co.,Ltd., Tokyo, JAPAN (non-U.S. corporation)			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 7049079	B1	20060523	
	WO 2001021205		20010329	
APPLICATION INFO.:	US 2000-831951		20000803	(9)
	WO 2000-JP5210		20000803	
			20010814	PCT 371 date
	NUMBER		DATE	
PRIORITY INFORMATION:	JP 1999-264687		19990917	
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	GRANTED			
PRIMARY EXAMINER:	Nolan, Patrick J.			
LEGAL REPRESENTATIVE:	White, Jr., Paul E., Manelli Denison & Selter PLLC			
NUMBER OF CLAIMS:	11			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	25 Drawing Figure(s); 25 Drawing Page(s)			
LINE COUNT:	1391			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methods of measuring the presence/absence of a coupling factor 6, which is a subunit of H.sup.+-transporting ATP synthase→H.sup.+ ATP synthase present in the mitochondrial inner membrane, in the blood and the concentration thereof are provided. Further, relations among the coupling factor 6 level in the blood and diseases and relations among the inhibition of the effect of the coupling factor and therapeutic effects on diseases are clarified and thus techniques for diagnosing and treating these diseases are provided.

The present invention provides a vector containing a DNA encoding the

coupling factor 6 or fragment thereof; a transformant transformed by this vector; and a method of producing the coupling factor 6 and its fragment. The present invention further provides an antibody reacting specifically with the coupling factor 6; a process of producing the antibody; and a method of assaying the coupling factor 6.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d his

(FILE 'HOME' ENTERED AT 11:21:27 ON 30 OCT 2008)

FILE 'REGISTRY' ENTERED AT 11:21:51 ON 30 OCT 2008
E "OLEYLOXYETHYLPHOSPHORYLCHOLINE"/CN 25
E "OLEYLOXYETHYLPHOSPHORYLCHOLINE"/CN 25
E "OLEYLOXYETHYL"/CN 25
L1 1 S 96720-06-8/RN

FILE 'MEDLINE, CAPLUS, WPIDS, USPATFULL' ENTERED AT 11:24:12 ON 30 OCT 2008
L2 27 S L1 OR OLEYLOXYETHYLPHOSPHORYLCHOLINE OR OLEYLOXYETHYLPHOSPHOC

=> s l2 and alzheimer
L3 0 L2 AND ALZHEIMER

=> s l2 and alzheimer's
MISMATCHED QUOTE 'ALZHEIMER'S'
Quotation marks (or apostrophes) must be used in pairs,
one before and one after the expression you are setting
off or masking.

=> s l2 and vasoactivity
L4 1 L2 AND VASOACTIVITY

=> d l4

L4 ANSWER 1 OF 1 WPIDS COPYRIGHT 2008 THOMSON REUTERS on STN
AN 2000-171073 [15] WPIDS
DNC C2000-053194 [15]
TI Modifying vasoactivity and inflammatory actions in microglia and neurons comprises regulating a soluble alphabeta pro-inflammatory pathway, useful for treating vascular diseases, e.g. cerebral amyloid angiopathy and vascular amyloidosis
DC B04
IN MULLAN M J; PARIS D; TOWN T C
PA (UYSF-N) UNIV SOUTH FLORIDA
CYC 84
PI WO 2000002561 A1 20000120 (200015)* EN 77[29]
AU 9949948 A 20000201 (200028) EN
ADT WO 2000002561 A1 WO 1999-US15947 19990713; AU 9949948 A AU 1999-49948 19990713
FDT AU 9949948 A Based on WO 2000002561 A
PRAI US 1998-92570P 19980713
IPCR A61K0031-00 [I,A]; A61K0031-00 [I,C]; A61K0031-121 [I,A]; A61K0031-121 [I,C]; A61K0031-165 [I,A]; A61K0031-165 [I,C]; A61K0031-18 [I,A]; A61K0031-18 [I,C]; A61K0031-21 [I,C]; A61K0031-27 [I,A]; A61K0031-352 [I,A]; A61K0031-352 [I,C]; A61K0031-357 [I,C]; A61K0031-36 [I,A]; A61K0031-366 [I,A]; A61K0031-366 [I,C]; A61K0031-403 [I,C]; A61K0031-4045 [I,A]; A61K0031-405 [I,A]; A61K0031-415 [I,A]; A61K0031-415 [I,C]; A61K0031-4427 [I,C]; A61K0031-4439 [I,A]

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=> d his

     (FILE 'HOME' ENTERED AT 11:21:27 ON 30 OCT 2008)

     FILE 'REGISTRY' ENTERED AT 11:21:51 ON 30 OCT 2008
                E "OLEYLOXYETHYLPHOSPHORYLCHOLINE"/CN 25
                E "OLEYLOXYETHYLPHOSPHORYLCHOLINE"/CN 25
                E "OLEYLOXYETHYL"/CN 25
L1            1 S 96720-06-8/RN

     FILE 'MEDLINE, CAPLUS, WPIDS, USPATFULL' ENTERED AT 11:24:12 ON 30 OCT
     2008
L2           27 S L1 OR OLEYLOXYETHYLPHOSPHORYLCHOLINE OR OLEYLOXYETHYLPHOSPHOC
L3            0 S L2 AND ALZHEIMER
L4            1 S L2 AND VASOACTIVITY

=>

---Logging off of STN---



=>
Executing the logoff script...


=> LOG Y

COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                      80.94      84.99

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                         ENTRY    SESSION
CA SUBSCRIBER PRICE                                      -9.60      -9.60

STN INTERNATIONAL LOGOFF AT 11:30:00 ON 30 OCT 2008
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